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Original Article

Correlation Analysis Between Serum Bile Acid Profiles and Colonic Neoplasms

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Abstract

Background and objectives: Currently, the mechanism of occurrence and development of colonic polyps and colonic cancer has not been fully elucidated. Previous studies have shown a certain relationship between bile acid (BA) profile and the development of colonic cancer. Through an analysis of the relationship between alterations in the serum BA profile and colonic neoplasms, this study sought to develop new biomarkers for assessing the risk of colon illnesses and offer fresh perspectives for identifying treatment targets.

Methods: The study encompassed 135 individuals who showed no abnormalities during colonoscopy, 204 patients with colonic polyps, and 92 patients with colonic cancer, all diagnosed and treated at Zhongda Hospital, Southeast University, from January 1, 2022, to June 1, 2023. Serum BA profiles, liver function, and clinical data were collected for statistical analysis.

Results: The concentration of deoxycholic acid in patients with colonic neoplasms was lower than that in the control group, whereas levels of taurocholic acid, taurochenodeoxycholic acid, and glycochenodeoxycholic acid were significantly higher in the colonic neoplasms group than in the control group ($P < 0.05$). Subgroup analysis revealed that there were statistical differences in the content of cholic acid, ursodeoxycholic acid, and glycoursodeoxycholic acid among different pathological types of colonic neoplasms. Logistic regression analysis indicated a negative correlation between the concentration of glycodeoxycholic acid and the risk of developing colonic neoplasms.

Conclusions: Compared with the normal population, the serum BA profile of colonic neoplasms patients has changed. Patients with colonic neoplasms exhibit elevated levels of primary conjugated BAs and decreased levels of secondary free BA (deoxycholic acid).

Introduction

Colonic cancer ranks second in the world's cancer fatality rates and is the third most prevalent malignancy worldwide.[1] One of the most prevalent precancerous lesions in colonic cancer is colonic polyps, which typically do not exhibit any noticeable clinical symptoms when they first appear.[2] They are generally identified during colonoscopy, and even some asymptomatic adenomatous polyps have already revealed focal canceration during pathological investigation.[3] The progression from healthy intestines through colonic polyps to colonic cancer represents the pathophysiological trajectory of colonic neoplasms. Clinicians are increasingly focusing on adenomatous polyps, which represent 50% to 70% of cases in colonic cancer patients.[4,5] Early detection and management of colonic polyps can markedly decrease the incidence of colonic cancer. Currently, the underlying mechanisms of colonic polyps and cancer development are not fully elucidated. Over the past decade, research has highlighted bile acids (BAs) as critical signaling molecules in the genesis of colonic neoplasms.[6] It is yet unknown, nevertheless, how colonic neoplasms and the serum BA profiles are related and how the disease develops.[7] This study compared the levels of 15 serum BAs between colonic neoplasm patients

Keywords: Serum; Bile acid profile; Colonic neoplasms; Colonic polyps; Colonic cancer; Bile acid metabolism; Deoxycholic acid; DCA; Primary conjugated BAs.

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase; BA, bile acid; BMI, body mass index; CA, cholic acid; CDCA, chenodeoxycholic acid; DCA, deoxycholic acid; GCA, glycocholic acid; GCDCA, glycochenodeoxycholic acid; GDCA, glycodeoxycholic acid; GLCA, glycolithocholic acid; GUDCA, glycoursodeoxycholic acid; LCA, lithocholic acid; TC, total cholesterol; TCA, taurocholic acid; TCDCA, taurochenodeoxycholic acid; TDCA, tauroursodeoxycholic acid; TLCA, taurolithocholic acid; TUDCA, tauroursodeoxycholic acid; UDCA, ursodeoxycholic acid.

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and healthy individuals and preliminarily explored the changes in serum BA profiles of colonic neoplasm patients. It analyzed the relationship between BA profiles and the development of colonic neoplasms, in order to find new biomarkers for evaluating disease risk and provide new directions for discovering therapeutic targets.

Materials and methods

Research object

The study conducted a retrospective analysis on 204 patients with colonic polyps and 92 patients with colonic cancer who received treatment at the Zhongda Hospital Southeast University between January 1, 2022, and June 1, 2023. They were chosen and included in the colonic neoplasms group based on a review of the electronic case system. The patients had an average age of (56.82 ± 8.74) years, with 160 males and 136 females. Inclusion criteria: (a) The study participants between the ages of 30 and 75 who have had a colonoscopy and received a pathology diagnosis of polyps or adenocarcinoma; (b) One week before their colonoscopy, they finished their serum bile acid profiles and liver function testing. Exclusion criteria: (a) Previous history of inflammatory bowel disease; (b) Previous liver and biliary system diseases, such as autoimmune hepatitis, sclerosing cholangitis, viral liver disease, cirrhosis, etc; (c) Previous intestinal surgery (excluding appendectomy); (d) Severe cardiopulmonary and renal dysfunction; (e) Patients with other malignant tumors; (f) Patients who have received chemotherapy or immunotherapy; (g) Pregnant women. Healthy people (135) with colonoscopies performed at our facility simultaneously comprised the control group; their inspection findings showed no appreciable abnormalities. They had an average age of (55.35 ± 8.79) years, with 61 men and 74 women. The exclusion criteria are the same as those for the colonic neoplasms group. This study was reviewed and approved by the Zhongda Hospital Institutional Review Board (Approval No.2021ZDSYLL297-P01). All procedures were carried out in accordance with the ethical guidelines of the Helsinki Declaration (as revised in 2013). The individual consent for this retrospective analysis was waived.

Research methods

Clinical information on study participants, including age, gender, body mass index (BMI), liver function, serum BA profiles, and tumor pathological parameters specific to the colonic neoplasms group, was collected. Liver function and BA profiles were assessed in all serum samples obtained during fasting.

Determination of serum BA profile

High-performance liquid chromatography-tandem mass spectrometry was used to identify 15 different forms of BAs in the BA profile, including (a) primary free BAs: cholic acid (CA) and chenodeoxycholic acid (CDCA), (b) primary conjugated BAs: taurocholic acid (TCA), glycocholic acid (GCA), taurochenodeoxycholic acid (TCDCA), and glycochenodeoxycholic acid (GCDCA), (c) secondary free BAs: deoxycholic acid (DCA), ursodeoxycholic acid (UDCA), and lithocholic acid (LCA); (d) secondary conjugated BAs: tauroursodeoxycholic acid (TDCA), glycodeoxycholic acid (GDCA), tauroursodeoxycholic acid (TUDCA), glycoursodeoxycholic acid (GUDCA), taurolithocholic acid (TLCA), and glycolithocholic acid (GLCA).

Sample processing for BA detection

Take 100 μL plasma samples, add 25μL BA deuterated internal standard solution and 200 μL acetonitrile methanol mixed solution successively, mix well, centrifuge at 4°C for 10 minutes at 14,000 r/min, and aspirate 150 μL supernatant for testing.

Liquid chromatography conditions

The chromatography column employed an Eclipse Plus C18 analytical column (100 × 3.0 mm, 3.5 μm). Mobile phase A consisted of a formic acid aqueous solution (*p*H = 3.25), and mobile phase B was a solution containing acetonitrile (acetonitrile: methanol = 80:20). The injection volume ranged from 1–10 μL (adjusted according to the analysis and instrument sensitivity). The column temperature was set at 40°C, utilizing gradient elution.

Mass spectrometry parameters

The ion source is an electric spray ion source, the scanning mode is a negative ion mode, the acquisition mode is a multi-reaction monitoring mode, the ion source temperature is 550°C, and the ionization voltage is −4,500 V.

Statistical analysis

Software called SPSS 26.0 was used to perform statistical analysis. Perform normality and homogeneity of variance tests on measurement data. The measurement data of normal distribution is represented by mean ± standard deviation, and an independent sample *t*-test is used to compare the two groups. The median and interquartile spacing [M (P25, P75)] are used to depict the measurement data of a skewed distribution. When comparing two groups, the independent sample non-parametric Mann-Whitney *U* test is employed; the Kruskal-Wallis H rank sum test is utilized for comparisons among several groups. The chi-square test is used to analyze and count data between groups. For correlation analysis, the Spearman rank correlation method is employed. The association between BA profiles and the risk of colonic neoplasm incidence is analyzed using binary logistic regression. $P < 0.05$ indicated that the difference was statistically significant.

Results

Comparison of the general situation of research subjects

A total of 135 people made up the control group, and 296 people with colonic neoplasms were included in this retrospective analysis. Regarding age, BMI, gender, ALT, AST, and TC, there was no statistically significant difference ($P > 0.05$) between the two groups, suggesting comparability (Table 1).

Comparison of serum BA compositions between colonic neoplasms group and control group

There was a statistical difference between the two groups in TCA, GCDCA, TCDCA, and DCA ($P < 0.05$). While the content of DCA was lower than that of the control group, the content of TCA, GCDCA, and TCDCA was significantly higher in the colonic neoplasms group. There was no statistical difference ($P > 0.05$) in the other BA components between the two groups, as shown in Table 2.

Comparison of serum BA profiles in patients with different pathological types of colonic neoplasms

For subgroup analysis, individuals with colonic neoplasms were categorized into three groups based on their pathological types: the non-adenomatous polyp group (59 cases), the adenomatous polyp

Table 1. Comparison of general conditions between the colonic neoplasms group and the control group

General information	Colonic neoplasms group (n = 296)	Control group (n = 135)	*P* value
Age (year)	56.82 ± 8.74	55.35 ± 8.79	0.107
BMI (kg/m^2)	23.98 ± 2.44	23.52 ± 2.37	0.068
Gender, n (%)			0.088
Males	160 (54.05%)	61 (45.19%)	
Females	136 (45.95%)	74 (54.81%)	
ALT (U/L)	17.00 (13.00, 24.75)	17.00 (13.00, 24.00)	0.853
AST (U/L)	20.00 (16.00, 24.00)	19.00 (16.00, 23.00)	0.351
TC (mmol/L)	4.38 ± 0.88	4.56 ± 0.91	0.062

Reference value range: ALT 9–50 U/L;AST 15–40 U/L;TC 0.00–6.20 mmol/L. ALT, alanine transaminase; AST, aspartate transaminase; BMI, body mass index; TC, total cholesterol.

group (145 cases), and the colonic cancer group (92 cases). As indicated in Table 3, there was no statistical difference in the compositions of BAs across the groups, except for statistical differences in CA ($P = 0.011$), UDCA ($P = 0.009$), and GUDCA ($P = 0.050$) among the various pathological types of colonic neoplasms. The content of CA was higher in non-adenomatous polyp group than in adenomatous polyp group ($P = 0.010$); the content of GUDCA was higher in non-adenomatous polyp group than in colonic cancer group ($P = 0.044$); the content of UDCA was higher in non-adenomatous polyp group than in adenomatous polyp group ($P = 0.011$); and the content of UDCA was higher in non-adenomatous polyp group than in colonic cancer group ($P = 0.022$). These findings were obtained through paired analysis on indicators with statistically significant differences.

Correlation analysis between serum BA profile levels and clinical pathological parameters of colonic neoplasms

As demonstrated in Table 4, no significant correlation was found between the levels of various serum BA profile components and tumor size within the colonic neoplasms group when these variables were analyzed using Spearman correlation analysis. However, there was a negative correlation between neoplasm patho-

Table 2. Detection results of serum BA profiles in the colonic neoplasms group and the control group (nmol /L)

BA components	Colonic neoplasms group (n = 204)	Control group (n = 135)	*P* value
Primary free BAs			
CA	65.60 (24.83, 234.00)	53.80 (27.60, 149.00)	0.362
CDCA	373.50 (94.90, 851.50)	294.00 (130.00, 625.00)	0.274
Primary conjugated BAs			
TCA	22.40 (5.80, 51.78)	12.70 (1.50, 32.30)	0.007*
GCA	159.00 (69.15, 321.75)	126.00 (52.90, 234.00)	0.057
GCDCA	900.00 (428.25, 1,810.00)	708.00 (298.00, 1,250.00)	0.008*
TCDCA	72.45 (27.15, 158.00)	41.60 (18.30, 119.00)	0.006*
Secondary free BAs			
DCA	146.00 (19.63, 434.00)	234.00 (82.60, 502.00)	0.009*
LCA	8.10 (0.40, 23.03)	6.40 (0.60, 21.00)	0.841
UDCA	67.50 (19.58, 230.75)	70.70 (19.00, 199.00)	0.802
Secondary conjugated BAs			
TDCA	8.15 (0.00, 30.05)	7.70 (0.00, 35.30)	0.827
GDCA	117.50 (11.90, 267.75)	125.00 (34.60, 335.00)	0.332
TLCA	0.15 (0.00, 2.48)	0.10 (0.00, 4.00)	0.299
GLCA	4.20 (0.00, 15.65)	5.40 (0.00, 18.10)	0.295
TUDCA	7.85 (3.325, 15.00)	8.20 (2.50, 15.00)	0.458
GUDCA	107.50 (42.78, 330.50)	122.00 (63.80, 283.00)	0.825

*$P < 0.05$, there is a statistical difference in this indicator between the two groups. BA, bile acid; CA, cholic acid; CDCA, chenodeoxycholic acid; DCA, deoxycholic acid; GCA, glycocholic acid; GCDCA, glycochenodeoxycholic acid; GDCA, glycodeoxycholic acid; GLCA, glycolithocholic acid; GUDCA, glycoursodeoxycholic acid; LCA, lithocholic acid; TCA, taurocholic acid; TCDCA, taurochenodeoxycholic acid; TDCA, tauroursodeoxycholic acid; TLCA, taurolithocholic acid; TUDCA, tauroursodeoxycholic acid; UDCA, ursodeoxycholic acid.

Table 3. Comparison of BA differences in different pathological types of colonic neoplasms (nmol/L)

BA components	Non-adenomatous polyp group (n = 59)	Adenomatous polyp group (n = 145)	Colonic cancer group (n = 92)	*H* value	*P* value
Primary free BAs					
CA	107.00 (39.50, 357.00)	53.20 (20.35, 185.50)	104.15 (26.60, 286.50)	9.075	0.011*
CDCA	408.00 (191.00, 1,130.00)	373.00 (80.50, 785.00)	298.50 (65.55, 999.75)	5.035	0.081
Primary conjugated BAs					
TCA	24.80 (8.90, 71.50)	20.10 (5.45, 45.80)	24.85 (7.50, 58.25)	1.937	0.380
GCA	160.00 (81.70, 423.00)	174.00 (72.60, 326.00)	132.50 (59.40, 283.75)	1.539	0.463
GCDCA	866.00 (458.00, 2,190.00)	961.00 (397.00, 1,785.00)	828.50 (409.50, 1,455.00)	1.560	0.458
TCDCA	113.00 (40.70, 185.00)	64.50 (24.60, 152.50)	70.65 (27.15, 132.25)	4.231	0.121
Secondary free BAs					
DCA	182.00 (38.50, 448.00)	118.00 (21.25, 401.00)	165.00 (7.18, 468.00)	1.208	0.547
LCA	5.30 (0.00, 16.80)	6.70 (0.50, 17.20)	12.65 (0.40, 26.83)	5.257	0.072
UDCA	107.00 (49.00, 311.00)	64.00 (16.15, 190.00)	50.00 (16.18, 238.25)	9.453	0.009*
Secondary conjugated BAs					
TDCA	17.90 (2.50, 39.10)	6.80 (0.00, 24.35)	7.95 (0.00, 26.63)	3.763	0.152
GDCA	135.00 (7.40, 398.00)	107.00 (19.25, 229.00)	132.50 (10.33, 353.00)	0.515	0.773
TLCA	0.00 (0.00, 2.10)	0.00 (0.00, 2.55)	0.50 (0.00, 2.68)	0.675	0.714
GLCA	4.40 (0.00, 20.30)	4.60 (0.00, 15.50)	3.90 (0.00, 13.58)	0.365	0.833
TUDCA	11.70 (4.50, 20.10)	6.50 (2.95, 15.00)	8.25 (3.50, 15.00)	5.398	0.067
GUDCA	220.00 (58.10, 543.00)	114.00 (43.55, 303.00)	81.65 (35.63, 280.75)	5.994	0.050*

*$P < 0.05$, there is a statistical difference in this indicator between the two groups. BA, bile acid; CA, cholic acid; CDCA, chenodeoxycholic acid; DCA, deoxycholic acid; GCA, glycocholic acid; GCDCA, glycochenodeoxycholic acid; GDCA, glycodeoxycholic acid; GLCA, glycolithocholic acid; GUDCA, glycoursodeoxycholic acid; LCA, lithocholic acid; TCA, taurocholic acid; TCDCA, taurochenodeoxycholic acid; TDCA, tauroursodeoxycholic acid; TLCA, taurolithocholic acid; TUDCA, tauroursodeoxycholic acid; UDCA, ursodeoxycholic acid.

logical type and the content of CDCA ($r = -0.121$, $P = 0.038$) and GUDCA ($r = -0.149$, $P = 0.010$).

Logistic regression model analysis of risk factors for colonic neoplasms

A univariate logistic regression analysis was conducted with the presence or absence of colonic neoplasms as the dependent variable and various other indicators as independent variables, aiming to investigate the risk factors for colonic neoplasms. Table 5 presents the findings, which indicated that CDCA (B = 0.000, OR = 1.000, $P = 0.049$), GCDCA (B = 0.000, OR = 1.000, $P = 0.043$), GDCA (B = 0.000, OR = 1.000, $P = 0.043$), and primary BA (B = 0.000, OR = 1.000, $P = 0.020$) were risk factors for colonic neoplasms formation and were connected with the risk of colonic neoplasms ($P < 0.05$). Multivariate logistic regression analysis was performed using indicators with statistically significant differences in univariate analysis. The findings indicated that GDCA (B = −0.001, OR = 0.999) was a protective factor for the development of colonic neoplasms.

Discussion

The occurrence and development of colonic neoplasms result from long-term evolution through comprehensive interactions between changes in genetic material and the external environment. Recent studies suggest that high concentrations of BAs may be known to induce cancer.[8] By triggering different signaling pathways in the body, BAs, as signaling molecules, can alter the colonic environment and regulate the development of colonic neoplasms.[9]

This study discovered that the colonic neoplasms group had a much higher amount of primary conjugated BAs (GCDCA, TCA, and TCDCA) than the control group ($P < 0.05$). A nested case-control analysis on 581 cases of primary colonic cancer identified between 1993 and 2008 was carried out in prior research by K ü hn T *et al*., who discovered a positive correlation between cancer risk and plasma levels of seven conjugated BA metabolites.[10] There are two secondary conjugated BAs, GDCA and TDCA, and five primary conjugated BAs, GCA, TCA, GCDCA, TCDCA, and GHCA. Consistent with the prior research, this study's findings suggest that elevated levels of primary conjugated BAs may be associated with an increased risk of colonic cancer. Experts and academics now widely agree that DCA plays a role in the onset and progression of colonic neoplasms. Numerous investigations have demonstrated a connection between colonic neoplasms and elevated fecal DCA levels.[11,12] It is yet unknown whether variations in serum DCA levels and the emergence of colonic neoplasms are related.

Moreover, it is still unclear if the patterns of DCA alterations in a person's serum and feces are the same. The study findings pre-

Table 4. Correlation analysis between serum BA profiles levels and clinical pathological parameters of colonic neoplasms

BA components	Size of tumor		Pathological type	
	r value	*P* value	*r* value	*P* value
CA	0.011	0.852	−0.028	0.636
CDCA	−0.032	0.580	−0.121	0.038
DCA	0.008	0.891	−0.067	0.251
LCA	0.072	0.217	0.069	0.237
UDCA	0.030	0.602	−0.112	0.055
GCA	−1.000	0.085	0.025	0.672
GCDCA	−0.062	0.285	−0.050	0.394
GDCA	0.014	0.806	−0.034	0.557
GLCA	−0.024	0.680	−0.034	0.555
GUDCA	−0.015	0.797	−0.149	0.010
TCA	0.006	0.919	0.037	0.523
TCDCA	0.003	0.957	0.009	0.876
TDCA	0.005	0.929	−0.094	0.107
TLCA	0.020	0.732	−0.033	0.575
TUDCA	0.021	0.717	−0.082	0.159

BA, bile acid; CA, cholic acid; CDCA, chenodeoxycholic acid; DCA, deoxycholic acid; GCA, glycocholic acid; GCDCA, glycochenodeoxycholic acid; GDCA, glycodeoxycholic acid; GLCA, glycolithocholic acid; GUDCA, glycoursodeoxycholic acid; LCA, lithocholic acid; TCA, taurocholic acid; TCDCA, taurochenodeoxycholic acid; TDCA, tauroursodeoxycholic acid; TLCA, taurolithocholic acid; TUDCA, tauroursodeoxycholic acid; UDCA, ursodeoxycholic acid.

Table 5. Risk factors for colonic neoplasms: Univariate and multivariate logistic regression analysis

Variable	Univariate analysis		Multivariate analysis	
	OR (95%CI)	*P* value	OR (95%CI)	*P* value
CA	1.000 (1.000, 1.001)	0.158		
CDCA	1.000 (1.000, 1.000)	0.049	1.000 (0.999, 1.001)	0.878
DCA	1.000 (1.000, 1.000)	0.643		
LCA	1.000 (0.999, 1.001)	0.655		
UDCA	1.000 (1.000, 1.001)	0.363		
GCA	1.000 (1.000, 1.001)	0.397		
GCDCA	1.000 (1.000, 1.000)	0.043	1.000 (1.000, 1.001)	0.406
GDCA	1.000 (0.999, 1.000)	0.043	0.999 (0.998, 1.000)	0.001
GLCA	0.999 (0.998, 1.000)	0.193		
GUDCA	1.000 (1.000, 1.001)	0.311		
TCA	1.000 (0.998, 1.002)	0.771		
TCDCA	1.001 (0.999, 1.002)	0.270		
TDCA	0.998 (0.995, 1.001)	0.135		
TLCA	0.997 (0.987, 1.007)	0.556		
TUDCA	1.005 (0.993, 1.017)	0.396		
primary BA	1.000 (1.000, 1.000)	0.020	1.000 (1.000, 1.001)	0.720
primary free BA	1.000 (1.000, 1.000)	0.051		
primary conjugated BA	1.000 (1.000, 1.000)	0.092		
secondary BA	1.000 (1.000, 1.000)	0.429		
secondary free BA	1.000 (1.000, 1.000)	0.857		
secondary conjugated BA	1.000 (1.000, 1.000)	0.153		

BA, bile acid; CA, cholic acid; CDCA, chenodeoxycholic acid; DCA, deoxycholic acid; GCA, glycocholic acid; GCDCA, glycochenodeoxycholic acid; GDCA, glycodeoxycholic acid; GLCA, glycolithocholic acid; GUDCA, glycoursodeoxycholic acid; LCA, lithocholic acid; TCA, taurocholic acid; TCDCA, taurochenodeoxycholic acid; TDCA, tauroursodeoxycholic acid; TLCA, taurolithocholic acid; TUDCA, tauroursodeoxycholic acid; UDCA, ursodeoxycholic acid.

sented in this article suggest that patients with colonic neoplasms have lower serum levels of secondary free BA DCA. There is still no agreement regarding the pattern of alterations in other BA components in patients with colonic neoplasms.[13] The results of various research' detection and analysis varies significantly.[6] It is evident, therefore, that the serum BA profiles of patients with colonic neoplasms differ from those of normal individuals. These differences are somewhat associated with the onset and progression of colonic neoplasms. We should incorporate the patients' blood and fecal BA profiles in follow-up investigations and perform synchronous comparison analysis to better understand the role of BA profiles in colonic neoplasms.

After further categorizing and assessing the colonic neoplasms group based on pathological type, we discovered statistical variations in CA, UDCA, and GUDCA amongst neoplasms with various pathological types. In this study, the non-adenomatous polyp group had higher levels of UDCA and GUDCA than the adenomatous polyp group, which was followed by the colonic cancer group. We hypothesize that UDCA and GUDCA could offer novel targets and approaches for the prophylaxis and management of colonic neoplasms. A correlation analysis was performed between the levels of serum BA profile and the pathological parameters of colonic neoplasms. The results indicated that there was a negative link between the pathological types of the neoplasms and CDCA and GUDCA. We can draw the conclusion that the incidence and progression of colonic neoplasms are associated with the decline in CDCA and GUDCA levels. GDCA (B = −0.001, OR = 0.999) was identified as a protective factor for the development of colonic neoplasms through logistic regression analysis of the risk factors, but the correlation between GDCA and colonic neoplasms was not strong. The aforementioned results suggest that in clinical practice, we should actively advise patients with alterations in BA composition—particularly when UDCA, GUDCA, and CDCA levels fall—to enhance colonoscopy examination. Such improvements will enhance patient prognosis and alleviate the burden on survivors.

Previous studies have analyzed the role and mechanism of BA profiles in the occurrence and development of colonic neoplasms. The widely recognized view is that increasing the concentration of DCA in the BA profiles can promote the occurrence of colonic neoplasms, while increasing the concentration of UDCA may inhibit the occurrence and development of tumors.[14,15] Besides, research has found that through numerous mechanisms, including the induction of β-catenin signal transduction, the upregulation of Cyclin D1 expression, the degradation of p53, and the promotion of resistance to cell death, DCA can induce aberrant proliferation and malignant transformation of colon cells.[16,17] Giving DCA-rich meals to $Apc^{min/+}$ mice led to an increase in the size and quantity of adenomas in their intestines, as well as an increase in the adenoma adenocarcinoma sequence, according to research by Liu *et al*.[18] In the intestinal mucosal tissue of DCA-treated mice, cytoplasmic tight adhesin-1, intestinal cell count, and the amount of released immunoglobulin A were all shown to be significantly lower. The findings suggest that by controlling the intestinal barrier, DCA may facilitate the growth of intestinal neoplasms. Colonic cancer is thought to be prevented by UDCA.[19] Studies have revealed that UDCA possesses anti-inflammatory, anti-apoptotic, and antioxidant properties in mice's digestive systems.[20] In the AOM model of experimental mouse colonic cancer, Sharad Khare *et al*.[21] discovered that DCA significantly increases tumorigenesis, but UDCA can reduce AOM carcinogenesis by preventing DCA-induced p38 activation and lowering the overexpression of C/EBPβ and Cox-2. Furthermore, UDCA can prevent DCA-induced transcription factor activation of AP-1 and NF-κB.[22] Interventions targeting NF-κB and AP-1 may partially slow the growth of colonic cancer. This investigation has found that the content of UDCA changes in patients with different pathological types of colonic neoplasms. Further research is required to fully understand UDCA's utility in detecting and managing colonic cancer patients.

In conclusion, there is a significant correlation between the incidence and progression of colonic cancers and the level of BA profiles. The potential therapeutic targeting of different BA profile components for colonic cancers remains a subject of ongoing debate, necessitating more research. This work indicates that regulating the content and composition of serum BA, even in the stage of colonic polyps and in the absence of intestinal abnormalities, can somewhat inhibit polyp formation and prevent its progression into cancer. Furthermore, this study acknowledges certain limitations. Firstly, this study is retrospective, and various confounding factors, such as the inconsistency in the operator performing the colonoscopies among the study subjects, current gastrointestinal symptoms, past disease history, and other elements, may have impacted the research outcomes. Secondly, the scope of the research findings was limited, with the analysis confined to serum BA profiles. Future research could extend to obtaining both fecal and serum samples from participants, allowing for comparison and analysis of the BA profile compositions of the two to identify more distinctive biomarkers. For future experimental designs, improvements are necessary, including expanding the sample size, collecting fecal samples, and conducting multicenter studies in collaboration with other hospitals. These steps will provide evidence for identifying effective targets to reduce the production of colonic polyps and decrease the incidence of colonic cancer.

Conclusions

This study suggests a potential relationship between serum BA levels and the incidence and progression of colonic neoplasms. Patients with colonic neoplasms exhibited higher levels of primary conjugated BAs and lower levels of secondary free BAs. Further research is necessary, as the current understanding of how components of the BA profile are involved in colonic neoplasms remains unclear.

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Conflict of interest

There are no conflict of interests regarding the publication of this paper.

Author contributions

Contributed to study concept and design (XJ and HC), performance of experiments (XJ), analysis and interpretation of data (XJ and HC), manuscript writing (XJ), critical revision (XJ and HC), statistical analysis (XJ), technical or material support (HC).

Ethical statement

This study was reviewed and approved by the Zhongda Hospital Institutional Review Board (Approval No.2021ZDSYLL297-P01). All procedures were carried out in accordance with the ethical guidelines of the Helsinki Declaration (as revised in 2013). The individual consent for this retrospective analysis was waived.

Data sharing statement

Data supporting the research article are available from the corresponding author or first author upon reasonable request.

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